Title

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on body weight within a randomized diet trial[1]

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Abstract

We examined the effect of change in dietary energy density on body weight in participants of a randomized trial in which the intervention group markedly increased fruit and vegetable intake while reducing energy intake from fat. Participants were 2718 breast cancer survivors, aged 26-74 years, with a mean body mass index (BMI) of 27.3 kg/m^2 (SD=6.3) at baseline. We assessed dietary intake by sets of four 24-hour dietary recalls and validated with plasma carotenoid concentrations. Weight and height were measured at baseline, 1 year, and 4 years. Energy density of the diet was calculated using food but excluding beverages. Intervention participants significantly reduced their dietary energy density compared to controls and maintained it over 4 years – both in cross-sectional ($p < 0.0001$) and longitudinal (group by time interaction $p < 0.0001$) analyses. Total energy intake or physical activity did not vary between the groups. The intervention group had a small but significant weight loss at 1 year (group by time interaction $p < 0.0001$) but no between-group weight difference was observed at 4 years. Our study showed that reducing dietary energy density did not result in a reduction in total energy intake, and suggests that this strategy alone is not sufficient to promote long-term weight loss in a free-living population.

Key words: Energy density, energy intake, physical activity, body weight, clinical trial

Introduction

Fiber, water, and fat are the three most important determinants of dietary energy density (1-3). Consequently, most fruit and vegetables are generally low in energy density due to their high fiber and water content (4-7). It has been observed that the volume of an individual's dietary intake remains more or less constant (8), which has led to the hypothesis that people may regulate their food intake based on volume rather than total energy. Accordingly, replacing energy-dense high-fat foods with much less energy dense fiber-rich foods such as vegetables and fruit should result in a reduction of energy intake and weight loss (8,9).

Various cross-sectional studies have found that individuals who eat high-energy-dense foods consume more energy and are relatively heavier than those who consume proportionately greater amounts of low-energy-dense foods (10-13). A number of feeding studies that have manipulated dietary energy density suggest that a decrease in energy density is associated with weight loss (14,15). Although these feeding studies address important questions about the association between energy density and weight loss, longer-term studies of individuals eating in real life situations are necessary to test the hypothesis.

Ad-libitum randomized trials that have encouraged participants to increase their fruit and vegetable intake and/or decrease their fat intake have had mixed results in terms of the amount of validated dietary change as well as weight change (16-23). None of these trials reported the energy density of the diets in the intervention and control groups, and thus it is possible that those studies that did not observe a decrease in weight may not have achieved a significant change in dietary energy density.

This report investigates the relationship between change in dietary energy density and body weight as an ancillary report of the Women's Healthy Eating and Living (WHEL) Study -- a large-scale randomized trial of the role of a plant-based dietary pattern in reducing breast cancer recurrence and death (24). Participants in the WHEL Study intervention group significantly increased their fruit, vegetable, and fiber intake and decreased their intake of energy from fat (25,26), a pattern characterizing a low-energy density diet, while the control participants consumed their usual diet. The WHEL Study has assessed dietary intake at multiple time-points, and thus provides the necessary data to assess change in dietary energy density according to method reported in the literature (27). In this report, we compare dietary energy density between the intervention and the control groups at baseline and demonstrate the association between dietary energy density and body weight. Then, we investigate the relationship of change in energy density to change in weight between study groups up to 4 years post-randomization.

Materials and Methods

This report considers participants of the WHEL Study. Population characteristics, eligibility criteria, randomization procedures, and dietary intervention protocol have been described in detail elsewhere (24,26).

Population

All women enrolled in the WHEL Study who did not have a study endpoint (death or recurrence) by 4 years of follow-up were eligible for the present study (n = 2718). WHEL Study participants were aged 18-70 years at cancer diagnosis; treated for primary, operable, and invasive stage I, II, or IIIA breast carcinoma; and at study entry were not receiving or scheduled for chemotherapy and had no evidence of cancer recurrence after initial treatment. Enrollment in another dietary trial, pregnancy, receiving estrogen replacement therapy, and presence of life-threatening medical conditions or diseases were key exclusion criteria.

The present study used WHEL baseline, 1-year, and 4-year follow-up data and adopted its randomized design for data analysis (control = 1363, intervention = 1355). Dietary data at baseline, 1 year, and 4 years were available for 2713 (control = 1360, intervention = 1353), 2465 (control = 1270, intervention = 1195), and 2324 (control = 1202, intervention = 1122) women respectively. At the same time points, 2718 (control = 1363, intervention = 1355), 2306 (control = 1174, intervention = 1132), and 2146 (control = 1116, intervention = 1030) women had their body weight measured.

Informed written consent from study participants was collected in the WHEL Study. The Human Subjects Committee of the University of California, San Diego, and all participating institutions approved the study procedures.

Dietary intervention

Participants in the intervention group were encouraged to maintain a dietary pattern that included a daily consumption of at least 5 vegetable servings, 16 ounces of vegetable juice (or equivalent vegetable servings), 3 fruit servings, 30 grams of fiber (18 g/1000 kcal), and 15-20% energy from fat (24,26). Telephone counseling, monthly cooking classes, and newsletters were the principal methods to promote dietary change in the intervention participants. Control group participants received print materials that included dietary guidelines from the U.S. Department of Agriculture (28) and the National Cancer Institute (29) and a bimonthly cohort maintenance newsletter with general health and nutrition information unrelated to the intervention group's dietary goals.

Dietary assessment

Dietary intake was assessed through a set of four 24-hour dietary recalls at baseline, 1 year, and 4 years. Trained dietary assessors conducted these recalls by telephone on randomly selected days, stratified for weekend vs. weekdays, over a 3-week period. The Nutrition Data System for Research (NDS-R) software was used to collect and estimate dietary intakes (NDS-R version 6.0, 2006, University of Minnesota, Minneapolis, MN). NDS-R included more than 18,000 food codes, including many ethnic foods and over 8,000 brand-name products.

A number of strategies were used to maximize the accuracy of dietary recall data (30). Dietary assessors completed a training program that included standardized data collection, proper interview technique, and efficient use of dietary analysis software. Participants were trained, before study enrollment, to estimate serving sizes with food models, measuring cups and spoons, and were provided with two-dimensional food models for reference during recalls. In addition, assessors used a multi-pass method that improved recall accuracy by prompting to obtain detailed data about type, amount, and preparation method of foods eaten.

Calculation of dietary energy density

We determined a participant's dietary energy density (kcal/g) (1 kcal = 4.18 kJ) for a dietary recall day by estimating total energy intake (kcal) for that day and dividing it by the total amount (g) of food reported being consumed on that day. Energy density values of the set of 4 days were averaged to derive a mean dietary energy density value for each participant. In our calculations, we excluded all beverages.

Physical activity assessment

Physical activity was determined from the Personal Habits questionnaire developed for Women's Health Initiative (WHI) (31), expressed as metabolic equivalents per week (Met-min/week) (32), and completed at baseline, 1 year, and 4 years. For the WHEL Study, this questionnaire was calibrated with the standard 7-Day Physical Activity Recall (PAR) (33) and validated with an accelerometer reading (34). The accelerometer measured an average of 165 total minutes of physical activity per week, which was not statistically different from the 187 minutes reported for the PAR or the 171 minutes reported with the WHI 9-item questionnaire.

Ascertainment of body weight

Weight and height were measured—with the participants wearing light clothing and no shoes -- during clinic visits (baseline, year 1, and year 4) scheduled in the WHEL Study. Body mass index (BMI) was calculated as weight (kg)/height (m^2).

Other variables

Information on cancer stage (I, II, IIIA) and demography was ascertained through medical records and questionnaire respectively. Age at study entry was categorized into 10- year age groups (<44, 45-54, 55-64, and ≥ 65 years) and race was categorized as non-Hispanic white, African-American, Hispanic, Asian-American, and others. Other variables included were education (college-graduate vs. non-graduate), employment status (yes, no), marital status (married vs. not married), and smoking (current, past, and never). We calculated summary variables such as total fruit and vegetable intake (servings/d) and percent energy intake from fat/d from 24-hour dietary recalls.

Validation of dietary intake with biomarkers

Plasma carotenoids are well-known biomarkers of fruit and vegetable intake (35). The WHEL Study measured plasma carotenoid concentrations on a 28% random sample of participants identified at baseline and has published plasma carotenoid measurement procedures and baseline to 1-year results (25,36). In this analysis, we report total plasma carotenoid concentrations on the available population (n=881) at baseline, 1 year, and 4 years. Total plasma carotenoids are the sum of the individual carotenoid separated and quantified (α-carotene, β-carotene, β-cryptoxanthin, lycopene, and lutein plus zeaxanthin) using high-performance liquid chromatography methodology (25). The mean laboratory day-to-day coefficient of variation for total plasma carotenoids was less than 7%.

Statistical Analyses

We compared baseline characteristics of the control and the intervention groups, including demographic, behavioral, and cancer related variables, thought to be potential confounders of the relationship between dietary intake and weight, were examined in this respect.

Energy density was calculated using 'food only' values. We used baseline values to assess univariable associations of energy density with categories of age, race, and BMI; one-way ANOVA compared category means against a referent category. We also grouped participants into tertiles of baseline dietary energy density, calculated mean values of total energy intake, physical activity, and body weight for each tertile and compared tertiles, using the lowest tertile as referent. We then compared baseline dietary energy density between the control and the intervention group and graphed energy density in each study group at each time period.

We also computed and compared total energy intake, physical activity, and body weight values in each study group at baseline, 1 year, and 4 years, testing for group differences with t-tests.

Finally, we used mixed effect models to assess change in energy density, total plasma carotenoids, total energy intake, physical activity, and body weight over the study follow-up period. We chose mixed models as they are the best option available for correlated data and for data with random missing values. 'Unstructured' covariance provided the smallest Akaike's Criterion (AIC) value, and was used in the mixed models.

All calculations were performed using SAS version 9.1 (Cary NC: SAS Institute). All statistical tests were two-tailed with an alpha level of 0.05.

Results

Baseline characteristics did not differ significantly between the randomly assigned control and intervention groups (Table 1). Women were 26 to 74 years of age (mean age 53.4, standard deviation [SD] = 8.8). The mean BMI was 27.3 (SD = 6.3); and 57% were overweight or obese. Although predominantly non-Hispanic white (85%), the cohort also included a small but varied group of minority women (4% African American, 3% Asian-American, 5%, Hispanic, and 3% other ethnicities). Well-educated (college graduate (54%)) and predominantly employed (72%), 70% of the WHEL women were also married. Only a small percentage (<5%) was diagnosed with either stage IIIA cancer or was currently smoking. The mean energy intake and physical activity were 1717 kcal/d

(SD=407) [7184(1703) kJ/d] and 868 MET-min/week (SD=879), respectively (data not shown).

At baseline, energy density was inversely associated with age (p for trend: <0.0001) and directly associated with BMI (p for trend: <0.0001). Asian-American participants reported the highest intake of fruit and vegetables and the lowest energy intake from fat (data not shown), making the energy density of their diets significantly lower than any other racial/ethnic group (Table 2). We observed strong linear trends (p <0.0001) across tertiles of energy density, with energy intake and body weight having strong positive associations and physical activity having a strong negative association. Participants in the highest tertile of energy density consumed, on average, approximately 300 kcal/d (1 kcal = 4.18 kJ) more and performed 450 MET-min/week less physical activity than participants in the lowest tertile; mean body weight differed by 6.8 kg between these two tertiles (Table 3).

Mean dietary energy density did not differ between the intervention and the control participants at baseline, although we observed a significant difference in dietary energy density between groups at 1 year and 4 years (p-values <0.0001). At 1 year, the intervention group reported consuming a diet that was 25% less energy dense than their baseline diet. At 4 years, this difference was still highly significant but had declined to 15% (Figure 1). The multivariate analysis (Table 4) shows that these group differences in energy density were statistically significant at both1 year and 4 years (p-values for group by time interaction <0.0001).

Total plasma carotenoid concentration, corroborated the between-group differences in fruit and vegetable intake as assessed by 24-hour recall. In the validation sample(36), no significant differences were observed between groups at baseline, and carotenoid values in the control group were relatively unchanged at 1 year and 4 years. In contrast, total plasma carotenoid concentrations in the intervention group increased substantially, resulting in a 66% difference between groups at 1 year, and a 41% difference at 4 years (data not shown).

Data for energy intake, physical activity, and body weight are presented in Figure 1 and Table 4. At baseline, mean weight in the intervention group was slightly higher than the control group (+0.2%). At 1 year, weight in the control group increased by 0.71 kg whereas weight decreased by 0.05 kg in the intervention group, resulting in a mean weight in the intervention group that was 0.7% lower than that of the control group. The multivariate analysis identified this difference as statistically significant (group by time interaction: <0.0001). At 4 years, both groups had gained weight and the mean weight for the intervention group was 0.7% higher than that of the control group. The longitudinal analysis did not identify this as statistically significant (group by time interaction: 0.23).

Reported energy intake was essentially the same at baseline and 1 year, and there was a non-significant 1.4% difference between groups at 4 years. At baseline, the intervention group performed 5% less physical activity than the control group. While both groups reported increasing their physical activity, the intervention group performed 3.6% less physical activity than the control group at 1 year and 0.3% less at 4 years. This change in physical activity was borderline significant at 4 years (group by time interaction: $p = 0.04$).

Discussion

In this group of breast cancer survivors participating in a long-term dietary trial, we observed that an increase in fruit and vegetable intake and decrease in percent energy from fat resulted in a substantial decrease in dietary energy density that was not accompanied by weight loss. Specifically, intervention participants significantly increased their intake of fruit and vegetables (2.7 and 2.3 servings/d respectively at 1 and 4 years, data not shown) and decreased their percent energy intake from fat (5.7 % and 4.3% respectively at 1 and 4 years, data not shown). These dietary changes resulted in a large decrease in dietary energy density, compared to the control group, whose diets and energy density remained relatively unchanged.

At 1 year we observed a 25% between group difference in dietary energy density, which was associated with small (0.7%) difference in weight in the hypothesized direction; while significant, this weight loss was much less than meets general guidelines for successful weight change (37-39). However, the intervention group sustained their reduction of dietary energy density through 4 years, and this reduction was not associated with a maintained lower weight. Accordingly, the results of this study do not support the hypothesis that a major reduction in dietary energy density will independently result in weight loss.

A key component of the energy density–weight loss hypothesis is the assumption that people who adopt a low energy density dietary pattern will regulate their food intake by volume rather than by total energy. We did not observe this phenomenon in our study population. Despite a substantial increase in fruit and vegetable intake in the intervention group, their total energy intake did not change at either follow-up point. Likewise, we observed no meaningful difference in change of physical activity, a surrogate marker of energy expenditure, between the study groups. Thus, physical activity does not explain the finding of no difference in weight change between groups.

This study is one of the few to examine a longitudinal association between a change in dietary energy density and body weight. Our findings differ from the results of the two other trials in the literature, that examined this association (40,41). In both trials, weight loss was significantly correlated with decrease in dietary energy density. However, differences in the study population, intervention, and duration of follow-up between those two trials (40,41) and the present one are substantial. The intervention in PREMIER trial (41), involves many more components than the dietary intervention in our study. In addition to promoting high-fiber and low fat diet, it promoted weight loss, restriction of alcohol and sodium and promotion of physical activity. Unlike our study, both trials (40,41) focused on overweight or obese participants, setting up the possibility of a regression to the mean effect on weight. Further, participants in our study maintained their dietary pattern across 4 years allowing us to investigate the long term influence of such a dietary pattern.

All dietary studies need to address measurement error, and low-energy reporting is a concern, as several studies have observed higher frequency of low-energy reporting in their intervention groups (42-45). A related issue is whether intervention participants were more prone to bias, and reported eating more “socially desirable” foods such as vegetables and fruits or less fat than actually consumed, which would directly influence dietary energy density. Although differential underreporting and social desirability bias among intervention participants is possible, that could not explain the dietary difference observed between our study groups. Total plasma carotenoids — a biomarker of fruit and vegetable consumption —increased significantly among intervention participants throughout the follow-up period but remained unchanged in the control group (Table 4).

This study has a number of strengths, primarily, its randomized trial design, whereby randomization theoretically distributes all attributes of the study participants, both measured and unmeasured, evenly between the groups. Neither reported caloric intake nor physical activity expenditure were different between study groups at any time point. The huge difference achieved in dietary energy density was confirmed with the accepted biomarker of vegetables and fruit. Further, the study measured body weight and height, unlike many other studies that used self-reported weight and height (46-48). Hence, the accuracy of outcome measures was higher. Finally, the cross-sectional associations of dietary energy density described in this report are consistent with findings from previous studies (10,11,27,49).

However, this study was not a random sample of the population. WHEL participants were breast cancer survivors, generally white, highly educated, and predominantly employed, therefore these results may not be generalizable to the population at large. Follow-up measured weight data were not available for 10% of participants who did not attend clinic visits, however, this missing data did not differ between study groups (control = 9.9%, intervention = 11.2%). Finally, this study could not address the hypothesis of whether low energy density in conjunction with caloric restriction leads to long-term weight loss.

In summary, the intervention in this randomized trial significantly reduced dietary energy density and maintained this change over 4 years. This change in dietary pattern, was not associated with a change in energy balance (total energy intake versus expenditure), and it did not result in a meaningful change in weight in free-living individuals. As a strategy to specifically reduce total energy intake, reducing dietary energy density may be a useful component of weight management. However, changing this characteristic of the diet without a targeted reduction in energy intake does not appear to result in either reduced energy intake or weight loss.

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Table 1: Baseline characteristics of the control and intervention group

	Control (n = 1363) %	Intervention (n = 1355) %	p-value
Age at study entry (years)			
20-44	16.9	15.3	
45-54	41.5	43.0	0.56
55-64	30.2	29.5	
≥ 65	11.4	12.2	
Body mass index (kg/m^2)			
<25	42.7	42.3	
25 – 29.9	32.1	31.3	0.74
≥ 30	25.2	26.4	
Race/ethnicity			
White	85.4	85.5	
Black	3.7	3.7	
Asian	3.5	3.0	0.93
Hispanic	5.1	5.6	
Others	2.3	2.1	
College Graduate	53.9	55.3	0.47
Married	70.6	70.3	0.83
Employed	72.8	71.9	0.61
Current smoker	4.8	4.1	0.16
Stage III cancer	4.2	3.9	0.83
	Mean ± Se[§]	Mean ± Se[§]	
Energy intake (kcal/day)	1718 ± 11.2	1714 ± 10.9	0.76
Physical activity (Met-min/week)	901 ± 24.3	854 ± 24.3	0.18

[§]Se: standard error of the mean.

Mean and standard error for continuous variables and frequency for categorical variables are presented

Table 2: Dietary energy density[1] (food only) by baseline demographic characteristics; The Women's Healthy Eating and Living (WHEL) Study (n[*]=2713).

Variable	n	Baseline Dietary Energy Density (Food only) Mean ± SEM [2]	p-trend
Age (years)			
≤44 (ref) [3]	437	1.57 ± 0.02	
45-54	1148	1.51 ± 0.02 [b]	< 0.0001
55-64	810	1.44 ± 0.02 [b]	
≥ 65	318	1.41 ± 0.02 [b]	
Body Mass Index (kg/m^2)			
<25 (ref) [3]	1151	1.41 ± 0.01	
25 – 29.9	862	1.52 ± 0.01 [b]	< 0.0001
≥ 30	700	1.57 ± 0.01 [b]	
Race/Ethnicity			
Non-Hispanic white (ref) [3]	2318	1.48 ± 0.01	
African American	102	1.65 ± 0.04 [b]	Not applicable
Asian American	88	1.32 ± 0.04 [b]	
Hispanic	145	1.55 ± 0.03	
Others	60	1.45 ± 0.05	

[*] Intervention and control group combined
[1]24Hour dietary recalls were used to obtain dietary information via telephone interview
[2]SEM: standard error of the mean.
[3]One-way ANOVA examined group differences; significant differences are denoted with letter "b".

Table 3: Energy intake, physical activity, and body weight by tertile of baseline dietary energy density (food only); n*=2713.

	Baseline Dietary Energy density (Food only)		
	Bottom Tertile <1.29 kcal[1]/g	Middle Tertile 1.29 – 1.60 kcal[1]/g	Top Tertile ≥ 1.61 kcal[1]/g
	Mean ± SEM[2]	Mean ± SEM[2]	Mean ± SEM[2]
Total energy intake (kcal[1]/day)	1571 ± 12.9	1698 ± 12.9 [b]	1874 ± 12.6 [c]
Physical activity (METs/week)[3]	1101 ± 29.6	903 ± 29.4 [b]	637 ± 22.9 [c]
Body weight (kg)	70.1 ± 0.56	72.8 ± 0.55 [b]	76.9 ± 0.55 [c]

* Intervention and control group combined
Reference: Bottom tertile; values with different superscript letters are significantly different ($p<0.05$);
[1] 1 kcal = 4.18 kJ.
[2] SEM: standard error of the mean.
[3] METs: Metabolic Equivalent Tasks. Sum of METs assigned as: 2 METs per minute of casual strolling, 3 METs per minute of mild activity or average walking, 4 METs per minute of fast walking, 5 METs per minute of moderate activity, 6 METs per minute of very fast walking , 8 METs per minute of strenuous activity.

Table 4: Changes in energy density, total energy intake, physical activity, and body weight over the study follow-up period; The Women's Healthy Eating and Living (WHEL) Study.

Factor	Group	Baseline	Change: Year 1-Baseline	Change: Year 4-baseline
		Mean ± SEM[2]	Mean ± SEM[2]	Mean ± SEM[2]
Energy density (Food only)	Control	1.49 ± 0.01	-0.03 ± 0.01	0.05 ± 0.01
	Intervention	1.48 ± 0.01	-0.35 ± 0.01***	-0.22 ± 0.01***
Total plasma carotenoids (µmol/L)	Control	2.47 ± 0.04	-0.07 ± 0.03	-0.10 ± 0.04
	Intervention	2.40 ± 0.03	1.59 ± 0.05***	0.94 ± 0.06***
Energy intake (kcal[1]/day)	Control	1718 ± 11.2	-121 ± 10.7	-152 ± 12.2
	Intervention	1713 ± 10.9	-115 ± 11.5	-172 ± 16.6
Physical activity (METs/week)[3]	Control	901 ± 24.6	51.2 ± 21.5	24.6 ± 24.5
	Intervention	854 ± 24.3	78.2 ± 21.2	72.2 ± 26.8*
Body weight (kg)	Control	73.3 ± 0.5	0.71 ± 0.11	1.43 ± 0.20
	Intervention	73.3 ± 0.5	-0.05 ± 0.12***	1.77 ± 0.23

Mixed effect models were used to examine difference of change between groups from baseline. P-values (*<0.05 **<0.01 *** <0.0001) were computed for testing group by time interaction for each variable.

[1] 1 kcal = 4.18 kJ.

[2] SEM: standard error of the mean.

[3] METs: Metabolic Equivalent Tasks. Sum of METs assigned as: 2 METs per minute of casual strolling, 3 METs per minute of mild activity or average walking, 4 METs per minute of fast walking, 5 METs per minute of moderate activity, 6 METs per minute of very fast walking, 8 METs per minute of strenuous activity.

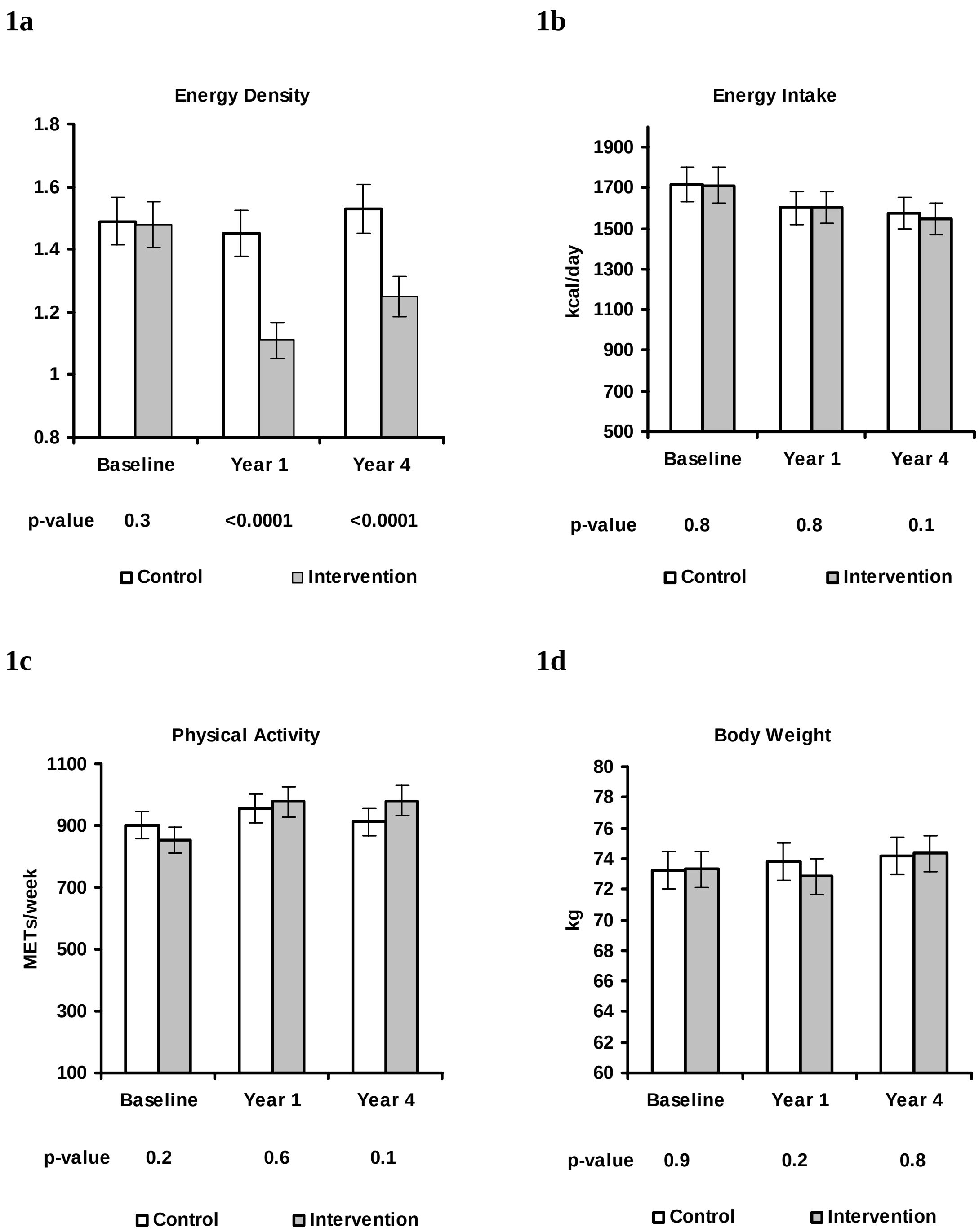

Figure 1a, 1b, 1c, and 1d

Mean energy density (food only), energy intake, physical activity, and body weight in the control and in the intervention group over the study period: The Women's Healthy Eating and Living (WHEL) Study; 1 kcal = 4.18 kJ.

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